failure.[2] Previous reports of permanent visual deterioration have all concerned large aneurysms of at least 12 × 15 mm.[23] The mechanism of visual loss has always been attributed to compression of the optic pathways either by aneurysm or by an associated haematoma with the pattern of loss being determined by the position of the aneurysm in relation to the optic nerves and chiasm.[4]

In this case, almost complete and permanent visual failure occurred six days after the primary haemorrhage. There was no evidence of further haemorrhage or clot on CT scans and the aneurysm was of moderate size with no signs of compression being seen at the time of surgery. The visual failure could have been produced by ischaemic damage to the anterior visual pathways secondary to vasospasm. A reduction in the cerebral blood flow over the period of onset of the visual failure was evident both clinically and by measurement.

Vasospasm has been postulated as a cause for transient ischaemic amaurosis before,[5] but this is the first time that such an ischaemic event has lead to permanent blindness and the vasospasm has been measured quantitatively during the evolution of the symptoms.

We suggest that ischaemia resulting from arterial spasm may play a role in the visual failure associated with anterior communicating artery aneurysms as well as direct compression of the visual pathways.

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Hemifacial spasm due to pontine infarction

Currently, hemifacial spasm (HFS) is thought to be due to a compression of the facial nerve at the root exit zone by blood vessels.[1] Compression by tumour, aneurysm or arteriovenous malformation has also been noted. We report a case probably due to a small infarct in the pons.

A fifty year old man, known to be hypertensive for 15 years, presented with a two year history of left hemifacial spasm. At the age of forty six, he had been admitted for a transient ischaemic episode probably in the right internal carotid territory. He recovered, but two years later, represented with a minor right hemiparesis and a transient left facial weakness. Two weeks later, he noted slight flutter-

Figure Magnetic resonance, T-1 weighted sagittal image. Low intensity signal in the pons.

ing of the lower eyelid which gradually increased in frequency and severity. The left hemifacial spasm was tonic, involved the orbicularis oculi, orbicularis oris and the platysma and was aggravated by light and emotional stress. The left limb reflexes were brisk. Therapy with botulinum toxin was effective.

The brainstem evoked potentials were impaired centrally on the left. Six months after the onset of the HSF, a blink reflex study[2] showed normal R1 (early component) latencies on both sides. The latencies of R2 (the late ipsilateral component) and R2c (the late contralateral component) were shorter on the left (28·7 and 30·1 ms) than on the right (33·4 and 35·1 ms) (control subjects: 33·2 + 2·7 and 34·8 + 2·9). With the paired stimuli technique, the R2 and R2c responses were obtained when the interstimuli interval was decreased to 100 ms on the left and to 200 ms on the right. In the control subjects, the inhibition of the R2 response was complete when the interstimuli interval decreased below 250 ms. The CT scan showed mild sub-cortical atrophy. MRI demonstrated an ectatic basilar artery and decreased signal on the T1-weighted MRI scan and increased intensity signal on the T2 weighted MRI scan in the right centrum ovale and in a small area just above and internal to the left facial nucleus (fig), suggesting a small infarct in the left pons.

To our knowledge, HFS due to a lacunar infarct has never been reported. The onset a few weeks after a regressive hemiparesis and the investigations support this view. The pathogenesis of HFS remains unclear. The ephaptic transmission hypothesis,[3] due to a compression by a blood vessel on the root entry zone, is widely popular. Our case agrees with the claim that the physiological abnormality is situated in the facial nucleus area and that signs of HFS are caused by hyperexcitability of the facial nucleus.[4] The results of the blink reflex of our patient, according to the study by Valls-Sole and Tolosa,[2] suggest an enhanced excitability of facial motor neurons and of those brainstem interneurons that mediate the blink reflex pathway. In our case, this hyperexcitability might not be due to antidromic impulses from compression of the root entry zone,[5] but to a loss of inhibitory impulses from the brainstem, due to the infarct in the pons.

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Suxamethonium is contraindicated in the Guillain-Barré syndrome

Suxamethonium induced hyperkalaemia has been described in a variety of disorders.[1] Ferguson *et al*[2] described four patients with chronic/relapsing polyneuropathy who developed life-threatening arrhythmias following suxamethonium administration. The presumed cause was suxamethonium induced hyperkalaemia although this was not documented in their patients. We have recently seen a patient with relapsing Guillain-Barré syndrome who developed severe ventricular arrhythmia secondary to a documented suxamethonium induced hyperkalaemia. The potential danger of the use of suxamethonium needs to be emphasised in the neurological literature.

A 51 year old man was admitted with a two week history of tingling in his hands and feet and progressive weakness in his arms and legs. These symptoms had begun one week after a flu-like illness. Examination revealed a proximal muscle weakness with depressed deep tendon reflexes and normal sensation. Cerebrospinal fluid examination was normal but nerve conduction studies showed evidence of a demyelinating neuropathy. A diagnosis of Guillain-Barré syndrome was made and he was treated with plasmapheresis with significant improvement over the following ten days. He was discharged but was readmitted two months later with an exacerbation of his symptoms.

Examination revealed severe weakness in his arms and legs, absent deep tendon reflexes, bilateral mild facial weakness, mild dysphagia and dysarthria. Forced vital capacity was two litres. He was treated with nasogastric feeding and daily plasmapheresis without any improvement over the following ten days. On day 11 because of deteriorating pulmonary function, it was decided to electively ventilate him. Before ventilation, there was a sinus tachycardia of 126/min but no other evidence of autonomic dysfunction. The arterial partial pressure of oxygen was normal and he was given 100% oxygen for three minutes before the procedure. Anaesthesia was induced with thiopentone and he was then paralysed with suxamethonium.

Immediately after the suxamethonium was given and before intubation he developed a ventricular tachycardia followed by a cardiac arrest. Cardio-pulmonary resuscitation was